CLAIMS

What is claimed is:

1. A method of inhibiting neural degeneration in a mammal, said method comprising reducing free iron levels in a neural tissue of said animal in an amount sufficient to inhibit neural degeneration in said neural tissue.

2. The method of claim 1, wherein said free iron levels are reduced by binding or chelating said iron by contacting said iron with an agent that binds or chelates iron.

3. The method of claim 2, wherein said agent is an iron-chelating small organic molecule.

4. The method of claim 3, wherein said iron-chelating molecule is a molecule selected from the group consisting of 5-chloro-7-iodo-8-hydroxyquinoline (clioquinol), deferiprone, desferrioxamine, pseudan, and derivatives thereof.

5. The method of claim 2, wherein said agent is systemically administered to said mammal.

6. The method of claim 2, wherein said agent is locally administered to a nerve tissue in said mammal.

7. The method of claim 9, wherein said agent is locally administered to brain tissue via a cannula.

8. The method of claim 2, wherein said agent is an iron-binding protein.

9. The method of claim 8, wherein said iron-binding protein is ferritin.

10. The method of claim 9, wherein said iron-binding protein is a ferritin heavy subunit (H ferritin).

11. The method of claim 8, wherein said iron-binding protein is recombinantly expressed.

12. The method of claim 8, wherein said iron-binding protein is recombinantly expressed *in vivo*.

13. The method of claim 8, wherein said iron-binding protein is recombinantly expressed in a nerve cell.

14. The method of claim 1, wherein said mammal is a human.

15. The method of claim 14, wherein said mammal is a human diagnosed as having or at risk for Parkinson's disease.

16. The method of claim 1, wherein said mammal is a non-human mammal.

17. The method of claim 1, wherein said neural tissue is brain tissue.

18. The method of claim 1, wherein said inhibiting neural degeneration comprises reducing dopaminergic cell loss.

19. A method of inhibiting the onset or progression of a disease characterized by neural degeneration in a mammal, said method comprising reducing free iron levels in a neural tissue of a mammal having or at risk for a disease characterized by neural degeneration.

20. The method of claim 19, wherein said disease is Parkinson's disease.

21. The method of claim 19, wherein said free iron levels are reduced by binding or chelating said iron by contacting said iron with an agent that binds or chelates said iron.

22. The method of claim 21, wherein said agent is an iron-chelating small organic molecule.

23. The method of claim 22, wherein said iron-chelating molecule is a molecule selected from the group consisting of clioquinol, deferiprone, desferrioxamine, pseudan, and derivatives thereof.

24. The method of claim 21, wherein said agent is an iron-binding protein.

25. The method of claim 24, wherein said iron-binding protein is ferritin.

26. The method of claim 25, wherein said iron-binding protein is a ferritin heavy subunit (H ferritin).

27. The method of claim 24, wherein said iron-binding protein is recombinantly expressed.

28. The method of claim 24, wherein said iron-binding protein is recombinantly expressed in vivo.

29. The method of claim 24, wherein said iron-binding protein is recombinantly expressed in a nerve cell.

30. The method of claim 19, wherein said mammal is a human.

31. The method of claim 30, wherein said mammal is a human diagnosed as having or at risk for Parkinson's disease.

32. The method of claim 19, wherein said mammal is a non-human mammal.

33. The method of claim 19, wherein said neural tissue is brain tissue.

34. The method of claim 19, wherein said inhibiting neural degeneration comprises reducing dopaminergic cell loss.

35. A method of mitigating one or more symptoms of a disease characterized by neural degeneration in a mammal, said method comprising administering to said mammal an agent that causes the sequestration or chelation of free iron in said mammal in an amount to mitigate one or more symptoms of said disease.

36. The method of claim 35, wherein said disease is Parkinson's disease.

37. The method of claim 35, wherein said administering comprises administering an iron chelator to said mammal.

38. The method of claim 37, wherein said iron-chelator is a molecule selected from the group consisting of clioquinol, deferiprone, desferrioxamine, pseudan, and derivatives thereof.

39. The method of claim 35, wherein said administering comprises administering an agent that upregulates expression of an endogenous iron chelator.

40. The method of claim 38, wherein said agent upregulates endogenous ferritin or hemoglobin.

41. The method of claim 35, wherein said administering comprises recombinantly expressing an iron-binding protein in a cell of said mammal.

42. The method of claim 41, wherein said iron-binding protein is ferritin.

43. The method of claim 41, wherein said iron-binding protein is a ferritin heavy subunit (H ferritin).

44. The method of claim 41, wherein said iron-binding protein is recombinantly expressed in vivo.

45. The method of claim 41, wherein said iron-binding protein is recombinantly expressed in a nerve cell.

46. The method of claim 19, wherein said mammal is a human.

47. The method of claim 19, wherein said mammal is a human diagnosed as having or at risk for Parkinson's disease.

48. The method of claim 19, wherein said mammal is a non-human mammal.

49. The method of claim 19, wherein said neural tissue is brain tissue.

50. The method of claim 19, wherein said inhibiting neural degeneration comprises reducing dopaminergic cell loss.

51. A method of inhibiting the onset or progression of a disease characterized by neural degeneration in a mammal, said method comprising reducing free iron levels in a neural tissue of a mammal having or at risk for a disease characterized by neural degeneration.

52. The method of claim 51, wherein said disease is Parkinson's disease.

53. The method of claim 51, wherein said free iron levels are reduced by binding or chelating said iron by contacting said iron with an agent that binds or chelates said iron.

54. The method of claim 53, wherein said agent is an iron-chelating small organic molecule.

55. The method of claim 54, wherein said iron-chelating molecule is a molecule selected from the group consisting of clioquinol, deferiprone, desferrioxamine, pseudan, and derivatives thereof.

56. The method of claim 53, wherein said agent is an iron-binding protein.

57. The method of claim 56, wherein said iron-binding protein is ferritin.

58. The method of claim 57, wherein said iron-binding protein is a ferritin heavy subunit (H ferritin).

59. The method of claim 56, wherein said iron-binding protein is recombinantly expressed.

60. The method of claim 56, wherein said iron-binding protein is recombinantly expressed in vivo.

61. The method of claim 56, wherein said iron-binding protein is recombinantly expressed in a nerve cell.

62. The method of claim 51, wherein said mammal is a human.

63. The method of claim 51, wherein said mammal is a human diagnosed as having or at risk for Parkinson's disease.

64. The method of claim 51, wherein said mammal is a non-human mammal.

65. The method of claim 51, wherein said neural tissue is brain tissue.

66. The method of claim 51, wherein said inhibiting neural degeneration comprises reducing dopaminergic cell loss.

67. A kit for mitigating the onset or progression of a disease characterized by neural degeneration in a mammal, said kit comprising:

an agent that increases sequestration or chelation of free iron in said mammal; and

instructional materials teaching the sequestration or chelation of free iron to mitigate the onset or progression of said disease.

68. The composition of claim 67, wherein said composition is formulated in a unit dosage formulation for mitigating the onset or progression of a disease characterized by neural degeneration in a human.

69. The composition of claim 67, wherein said disease is Parkinson's disease.

70. The composition of claim 67, wherein said agent comprises a nucleic acid that encodes a protein that chelates iron.

71. The composition of claim 70, wherein said protein is a ferritin.

72. The composition of claim 67, wherein said agent comprises an iron chelator.

73. The composition of claim 78, wherein said iron-chelator is a molecule selected from the group consisting of clioquinol, deferiprone, desferrioxamine, pseudan, and derivatives thereof.

74. A pharmaceutical composition for mitigating the onset or progression of a disease characterized by neural degeneration in a mammal, said composition comprising an agent that increases sequestration or chelation of free iron in said mammal.

75. The composition of claim 74, wherein said composition is formulated in a unit dosage formulation for mitigating the onset or progression of a disease characterized by neural degeneration in a human.

76. The composition of claim 74, wherein said disease is Parkinson's disease.

77. The composition of claim 74, wherein said agent comprises a nucleic acid that encodes a protein that chelates iron.

78. The composition of claim 77, wherein said protein is a ferritin.

79. In a mammal diagnosed as having or as at risk for a disease characterized by neural degeneration, a neural tissue in contact with an agent that chelates or sequesters free iron.

80. The neural tissue of claim 79, wherein said mammal is not diagnosed as having an iron overload disease.

81. The neural tissue of claim 79, wherein said agent is an iron chelator.

82. The neural tissue of claim 81, wherein said iron-chelator is a molecule selected from the group consisting of clioquinol, deferiprone, desferrioxamine, pseudan, and derivatives thereof.

83. The neural tissue of claim 79, wherein said agent is an iron-binding protein.

84. The neural tissue of claim 83, wherein said iron-binding protein is ferritin.

85. The neural tissue of claim 83, wherein said iron-binding protein is a ferritin heavy subunit (H ferritin).

86. The neural tissue of claim 83, n said iron-binding protein is recombinantly expressed.

87. The neural tissue of claim 83, wherein said iron-binding protein is recombinantly expressed in vivo.

88. The neural tissue of claim 83, wherein said iron-binding protein is recombinantly expressed in a nerve cell.

89. The neural tissue of claim 79, wherein said mammal is a human.

90. The neural tissue of claim 79, wherein said mammal is a non-human mammal.

91. The neural tissue of claim 79, wherein said neural tissue is brain tissue.

92. A method of evaluating the risk or progression of a disease characterized by neural degeneration in a mammal, said method comprising:

providing a biological sample from said mammal; and

determining the level of free iron in said sample where an elevated level of free iron as compared to that found in a sample from a normal healthy mammal indicates that said mammal is at risk for or progressing with said disease.

93. The method of claim 39, wherein said disease is Parkinson's disease.